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Original Article

Usefulness of combined CARTO electroanatomical mapping and manifest entrainment in ablating adenosine triphosphate-sensitive atrial tachycardia originating from the atrioventricular node vicinity

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ABSTRACT

Background: By using a noncontact mapping system, adenosine triphosphate (ATP)-sensitive atrial tachycardia (ATP-AT) originating from the atrioventricular (AV) node vicinity was successfully ablated at the entrance to the slow conduction zone indicated by the manifest entrainment technique. We aimed to prospectively validate the efficacy of the combination of CARTO electroanatomical mapping and manifest entrainment in ablating this ATP-AT.

Methods: Of the 27 AT patients from January 2013 to March 2014, 6 patients with sustained ATP-AT were studied (age, 67 ± 13 years; tachycardia cycle length, 350 ± 95 ms). We first created the CARTO map during AT, and performed rapid pacing from the anterior right atrial wall (ARAW) and cavotricuspid isthmus (CTI) approximately 30 mm remote from the earliest activation site (EAS). We identified the site where manifest entrainment, defined as the orthodromic capture of the EAS with a long conduction time, was observed, and ablated the site approximately 20 mm remote from the EAS, between the pacing site and the EAS.

Results: Manifest entrainment was demonstrated in all patients paced from the ARAW (four patients) and from the CTI (two patients). Ablation at the prespecified site terminated AT in 6 ± 3 s, and AT became no longer inducible in all patients. At the successful ablation sites, discrete atrial electrograms were recorded; however, low-amplitude, fractionated electrograms suggestive of slow conduction were not observed in all patients. The atrio-His interval during sinus rhythm remained unchanged (from 96 ± 12 to 89 ± 7 ms, p=NS). During 11 ± 6 months, no patients showed AT recurrence and AV conduction abnormality.

Conclusion: CARTO mapping- and manifest entrainment-guided ablation strategy is effective and safe in the treatment of ATP-AT.

1. Introduction

Entrainment pacing is useful not only in establishing reentry as a mechanism of tachycardia but also in identifying the pacing site relative to the slow conduction zone [1–3]. When manifest entrainment represented by constant fusion except for the last entrained beat in the electrocardiogram (ECG) is demonstrated, the pacing site is proximal to the slow conduction zone. When entrainment showing concealed fusion in the ECG, postpacing interval equal to the tachycardia cycle length and stimulus-to-QRS (P) equal to electrogram-to-QRS (P), is demonstrated, the pacing site is on the critical slow conduction zone [4–6]. This "concealed entrainment" is widely used as a tool to identify the effective ablation site in reentrant tachycardias.

Adenosine- or adenosine triphosphate (ATP)-sensitive atrial tachycardia (AT) (ATP-AT) with the earliest activation site (EAS) in the atrioventricular (AV) node vicinity was reported to be due to reentry [7], and catheter ablation performed for this specific AT has targeted the EAS with some risk of AV conduction impairment.

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ATP-AT was shown to have its origin not only in the vicinity of the AV node but also in other sites, including the coronary sinus ostium [8,9], tricuspid annulus [10,11], mitral annulus [12], and others [13]. By applying a single extrastimulus during ATP-AT originating from the AV node vicinity, Yamabe et al. demonstrated that verapamil- or ATP-sensitive atrial tissue not in the AV conduction system but close to the AV node forms the tachycardia reentry circuit [9]. More recently, while localizing the pacing site relative to the EAS during ATP-AT by using a noncontact mapping system (EnSite), Yamabe et al. demonstrated that this AT could be entrained by rapid pacing at a site remote from the EAS, fulfilling the classic or manifest entrainment criteria, and successfully ablated at the site that is supposed to be the entrance to the slow conduction zone [14]. This entrainment-based approach is rather different from those used previously [7–13], and therefore needs to be further validated. In the present study, we aimed to prospectively validate the efficacy of the combined use of a contact electroanatomical mapping system (CARTO) that is more accurate than the EnSite noncontact mapping system, and manifest entrainment in ablating this ATP-AT. We also sought whether any abnormal low-amplitude and fractionated electrograms, suggestive of slow conduction allowing small circuit reentry [15,16], were present particularly at the successful ablation site.

2. Material and methods

2.1. Study patients

Of the consecutive 27 patients with right AT, not including typical or atypical AV nodal reentrant tachycardia (AVNRT), and undergoing catheter ablation from January 2013 to March 2014, 7 (26%) had a diagnosis of ATP-AT with the EAS in the vicinity of the AV node. In one patient, AT was terminated during catheter mapping and sustained AT was no longer inducible. This study therefore included the successive six patients with sustained ATP-AT showing stable tachycardia cycle length, with variation < 15 ms. The patients comprised two men and four women, with a mean age of 67 ± 13 years (range, 55–83 years). Structural heart disease (dilated cardiomyopathy) was present in one patient.

In all study patients, a small dose of ATP (5 mg) terminated the tachycardia. AVNRT was excluded according to the following criteria [14]: (i) tachycardia induction was independent of the critical atrio-His (AH) interval prolongation; (ii) induction and perpetuation of tachycardia was independent of AV block; (iii) ventricular pacing delivered during tachycardia demonstrated AV dissociation without affecting the tachycardia cycle length; (iv) the His bundle (HB) electrogram was advanced by 15 ms by the ventricular pacing during tachycardia, with no change in the tachycardia cycle length; (v) spontaneous change in the HA interval with a fixed subsequent AA interval was observed; (vi) the difference between the AH interval during atrial pacing and that during tachycardia was < 20 ms; and (vii) a V-A-A-V sequence was observed when tachycardia was induced by premature ventricular stimulation.

2.2. Cardiac catheterization

The study protocol was in agreement with the guidelines for clinical studies delineated by the ethics committee of our institution. All patients gave written informed consent to participate in the electrophysiological study and catheter ablation. The study was done in the postabsorptive, nonsedated state. All antiarrhythmic drugs were discontinued for at least five half-lives before the procedure.

In all patients, a 7F 4-mm tip mapping catheter with an electrode spacing of 1–7–4 mm (Navistar; Biosense Webster, Diamond Bar, CA, USA) and a 5F quadripolar catheter (5–5–5 mm interelectrode spacing) (Inquiry catheter; St. Jude Medical, St. Paul, MN, USA) were introduced through the femoral vein and placed in the right atrium (RA) for mapping and at the HB region, respectively. The Navistar catheter was also used for programmed atrial stimulation from the high RA for the electrophysiologic study. A 5F decapolar catheter (Fepo; Fukuda Denshi Co., Ltd., Tokyo, Japan) was introduced through the internal jugular vein and inserted into the coronary sinus. A 7F steerable duodecapolar Halo catheter (Inquiry catheter, St. Jude Medical) was introduced through the femoral vein and positioned along the tricuspid annulus. All of the electrograms were recorded by using a digital multichannel system (ComboLab; GE Healthcare, Waukesha, WI, USA) along with the 12-lead ECG.

AT was induced by programmed atrial stimulation with the use of a cardiac stimulator (BC-1100, Fukuda Denshi Co., Ltd.). When sustained AT could not be induced, intravenous isoproterenol (0.5–1.0 μg/min) was continuously administered. In all study patients, electroanatomical CARTO mapping of the RA was performed during AT with the use of CARTO 3 system (Biosense-Webster, Diamond Bar, CA, USA).

2.3. Entrainment and ablation strategy

1. While referring to the RA activation map during AT, entrainment pacing was done during sustained AT for about 5 s at a rate 5–10 beats/min faster than the AT rate from the sites about 30 mm away from the EAS, including the anterior right atrial wall (ARAW), upper and lower interatrial septum, and cavotricuspid isthmus close to coronary sinus ostium (CTI-CS). For every attempt of entrainment pacing, we examined whether all of the atrial electrograms were captured at the pacing cycle length. If not, entrainment pacing was resumed while increasing the pacing rate by 5 beats/min.
2. Among the entrainment attempts from the four pacing sites, we identified the site at which pacing demonstrated manifest entrainment; manifest entrainment was defined by the orthodromic capture of the electrogram at the EAS with a long conduction time by the immediately previous pacing impulse $(N-1)$, and the antidromic capture of the other sites by the immediate impulse (N) in one paced beat except for the last entrained beat, equivalent to first entrainment criterion.
3. We then applied radiofrequency (RF) energy (20 W) at the site 15–20 mm remote from the EAS between the site showing manifest entrainment and the EAS. When AT was not terminated, the ablation site was moved by 2 mm laterally or toward the EAS, and RF energy was applied.
4. The end points of ablation was the termination of AT and the noninducibility of AT after isoproterenol infusion.

2.4. Characteristics of electrograms at the successful ablation site

Most of the reentrant circuits except for typical atrial flutter have a slow conduction zone, which is often represented by low-voltage, fractionated electrograms [15,16]. We therefore examined whether such abnormal electrograms were present at the successful ablation site.

2.5. Statistical analysis

Continuous variables are expressed as mean ± 1 standard deviation. Comparison of the AH intervals before and after ablation was done by using a two-tailed paired t test. A p-value of < 0.05 was considered statistically significant.

3. Results

The baseline characteristics of the study patients are shown in Table 1. The tachycardia cycle length was 350 ± 95 ms. To make the tachycardia sustained and stable, isoproterenol was infused in four patients. The activation map during AT was created by sampling from 152 ± 28 RA points, and it revealed a focal activation pattern with the EAS close to the HB recording site in all patients (Fig. 1A). The EAS during AT was 11 ± 7 mm above (n=3), below (n=1), posterior (n=1), or right (n=1) to the HB recording site. Four of the six patients had the EAS within 10 mm from the HB site.

3.1. Entrainment study

Manifest entrainment was demonstrated in all patients by the pacing from the ARAW in four patients and from the CTI-CS in the other two patients. The distance between the HB recording and pacing sites was 25 ± 4 mm (Table 1). In the two patients with the EAS close to the HB recording site and with manifest entrainment demonstrated from the CTI-CS, the V-A and AV intervals in the HB electrogram during the tachycardia were 145 and 150 ms, respectively, in patient 2, and 115 and 153 ms, respectively, in patient 3, indicating that common AVNRT was unlikely [17]. In all patients, the postpacing interval (PPI) was longer by ≥20 ms than the tachycardia cycle length, suggesting that the pacing site was outside of the reentry circuit.

Fig. 1 shows an example of entrainment of ATP-AT (cycle length, 420 ms) from the ARAW (pacing cycle length, 400 ms) (patient 1). The EAS (red tag) was 8 mm above the HB recording site (yellow tag), and the pacing site (blue tag) was 32 ms away from the EAS (panel A). Among the atrial electrograms recorded (panel B), that at the HB recording site was the earliest. The red and blue asterisks indicate the last electrograms captured by the last pacing stimulus. It was noted that the atrial electrograms at the HB recording site and recorded in Halo 1/2 to 5/6, and the

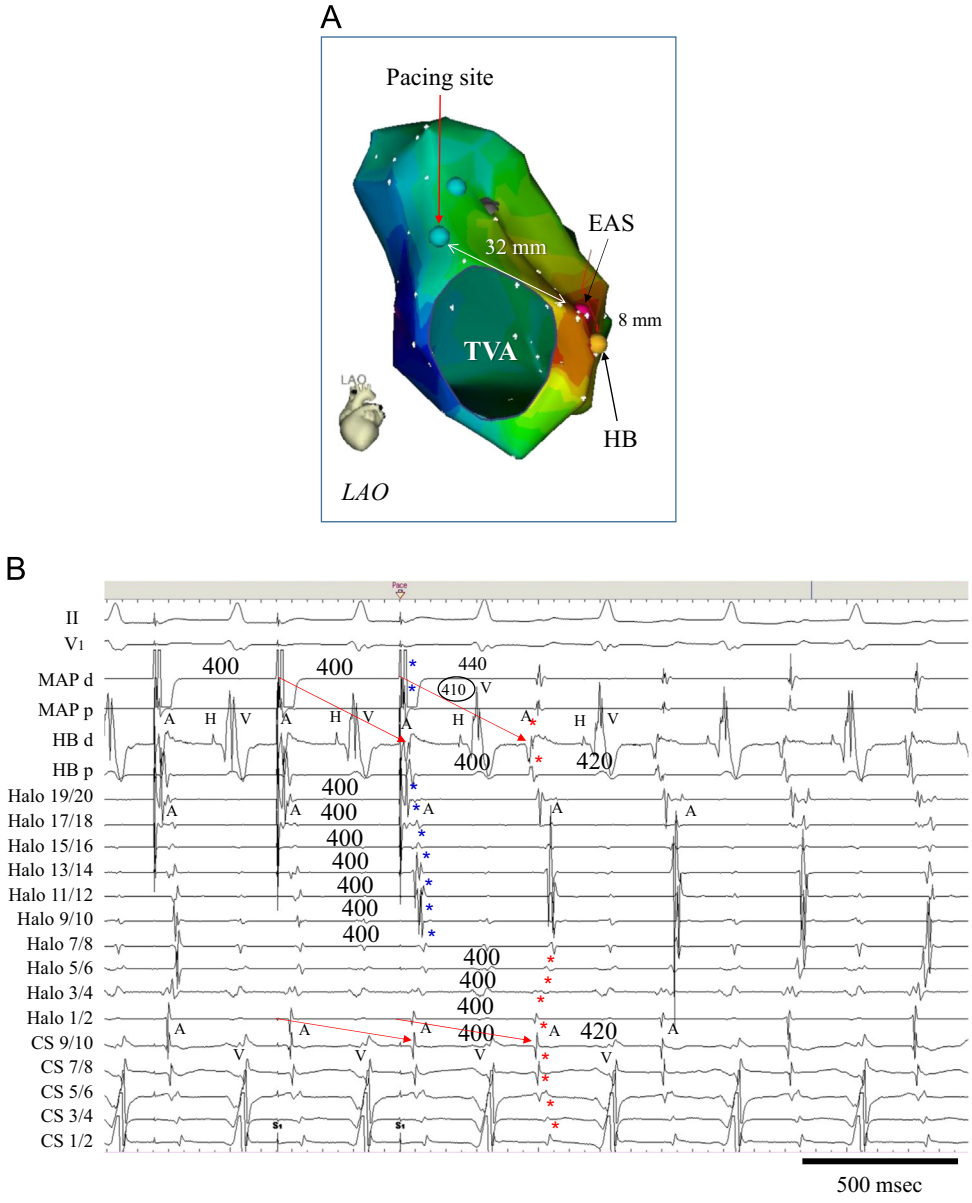

Fig. 1. *Panel A*, activation map during tachycardia (patient 1), showing a focal activation pattern with the earliest activation site (EAS) 8 mm above the His bundle (HB) recording site. *Panel B*, entrainment of tachycardia from the anterior right atrial wall (patient 1). The red and blue asterisks indicate the last electrograms captured by the last pacing stimulus. See text for discussion. MAP d and MAP p=distal and proximal pair of mapping catheter electrodes, respectively; HB d and HB p=distal and proximal pair of the HB recording catheter electrodes, respectively; Halo=each pair of Halo catheter electrodes; CS=each pair of coronary sinus catheter electrodes; TVA=tricuspid valve area; LAO=left anterior oblique projection.

Table 1
Baseline characteristics and entrainment study.

Pt no.	Age/sex	AT CL (ms)	ISP dose (μg/min)	EAS	Manifest entrainment		
					±	Site	Distance from the HB (mm)
1	57/F	420	–	8 mm above the HB	+	ARAW	32
2	55/F	295	0.5	6 mm below the HB	+	CTI-CS	25
3	55/M	268	1.0	12 mm above the HB	+	CTI-CS	26
4	83/F	511	0.5	9 mm posterior to the HB	+	ARAW	21
5	80/M	296	1.0	8 mm above the HB	+	ARAW	25
6	70/F	312	–	20 mm right to the HB	+	ARAW	37
Mean ± SD	67 ± 13	350 ± 95		11 ± 7 mm from the HB			25 ± 4

ARAW=anterior right atrial wall; AT CL=atrial tachycardia cycle length; CTI-CS=cavotricuspid isthmus close to coronary sinus ostium; EAS=earliest activation site; F=female; HB=His bundle recording site; ISP=isoproterenol; M=male; Pt=patient; SD=standard deviation.

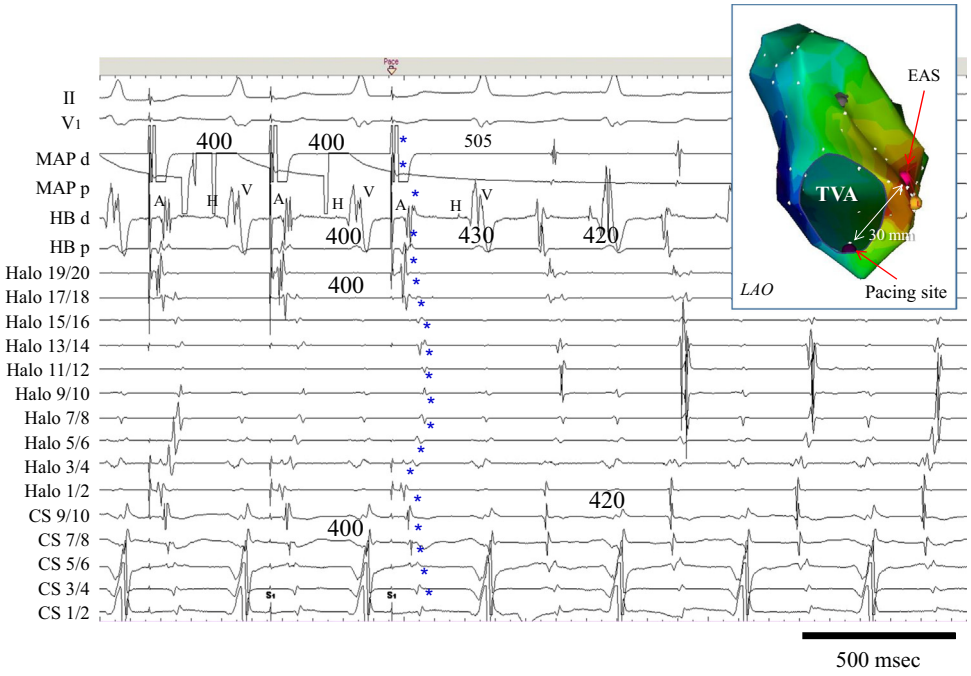

Fig. 2. Entrainment from a site in the cavotricuspid isthmus close to the coronary sinus ostium in the same case as in Fig. 1. Blue asterisks indicate the last electrograms captured by the last pacing stimulus. See text for discussion. All abbreviations are as in Fig. 1.

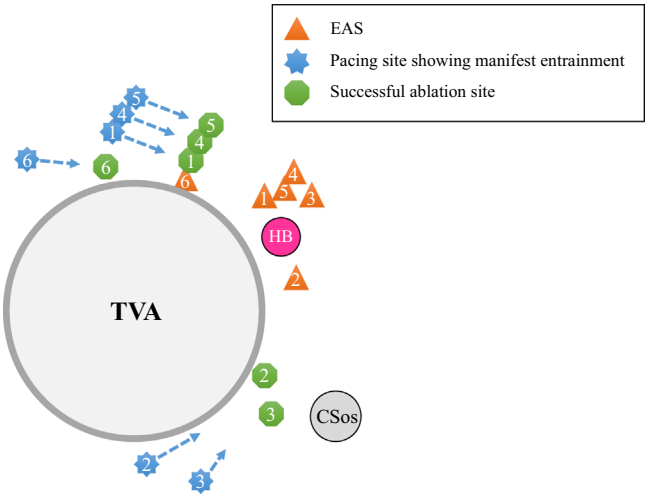

Fig. 3. Location of the earliest activation site (EAS), manifest entrainment site, and successful ablation site relative to the His bundle (HB) recording site for all six patients. CSos=coronary sinus ostium; TVA=tricuspid valve area.

coronary sinus (red asterisks) were captured orthodromically with a long conduction interval (red arrows) and with the same morphologies as during the tachycardia. The other electrograms (blue asterisks) were captured directly or antidromically, showing different morphologies from those during the tachycardia. Thus, manifest entrainment was demonstrated, indicating that this pacing site (ARAW) was proximal to the slow conduction zone.

Fig. 2 shows entrainment pacing from the CTI-CS in the same case as in Fig. 1, and all of the atrial electrograms were captured directly or antidromically (blue asterisks), showing different morphologies from those during the tachycardia. This indicates that the pacing site was distal to the slow conduction zone.

3.2. Catheter ablation

RF energy was applied during AT in all patients, except in one in whom it was during sinus rhythm because AT became unsustained after the entrainment study. Fig. 3 illustrates the location of the EAS, manifest entrainment site, and successful ablation site relative to the HB recording site for all six patients. In four patients, the ablation site

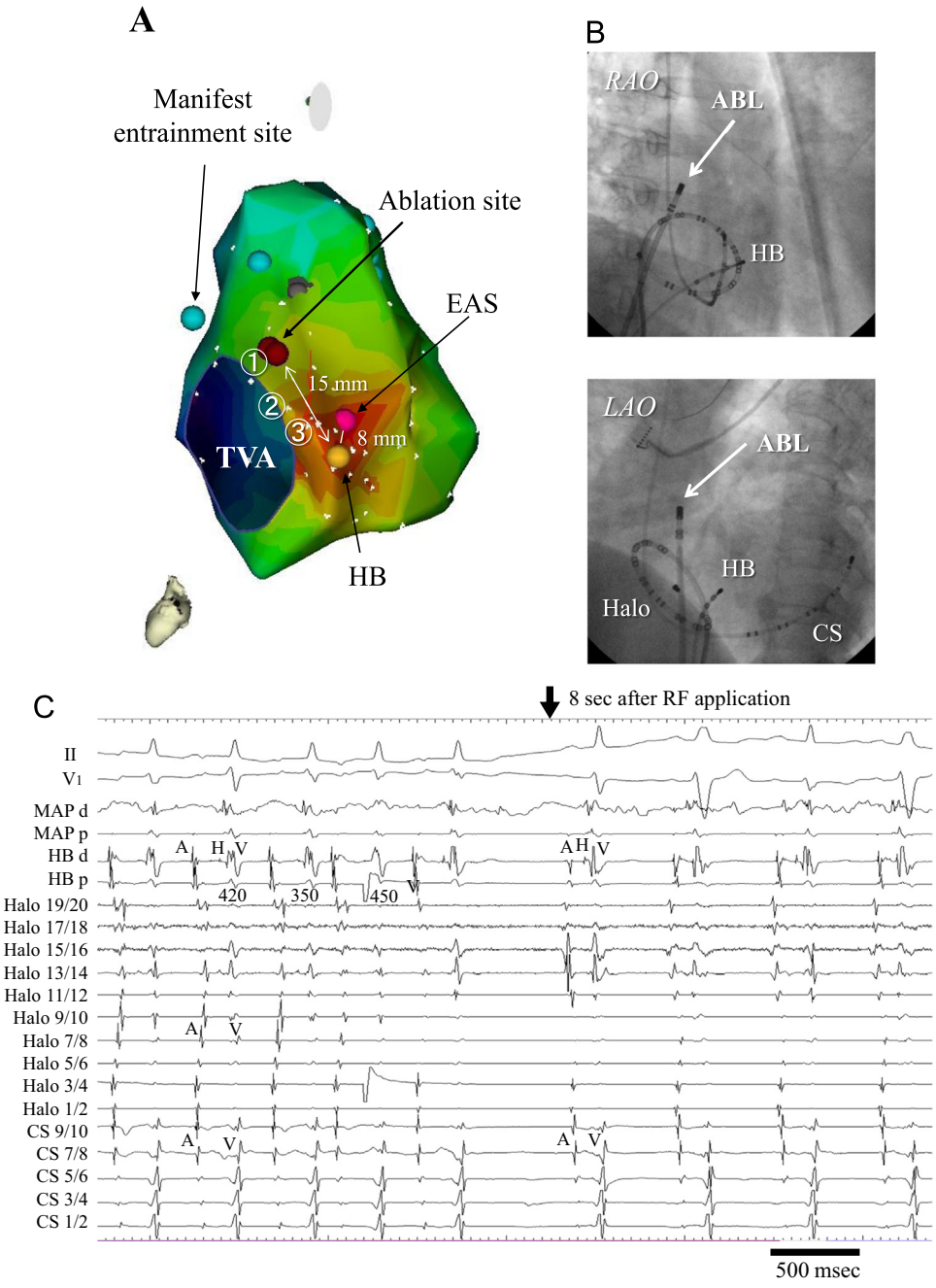

Fig. 4. *Panel A*, successful ablation site in the left lateral view (patient 1). It is located between the manifest entrainment pacing site and the His bundle (HB) recording site, and 15 mm from the HB site toward the pacing site. *Panel B,* fluoroscopy images showing the relative location of the ablation catheter (ABL) to the other catheters at the HB recording site, coronary sinus (CS), and Halo catheter in the right atrium. *Panel C*, termination of tachycardia after the application of radiofrequency (RF) energy. RAO = right anterior oblique projection; LAO = left anterior oblique projection. All other abbreviations are as in Fig. 1.

was in the anterior and right to the HB site. In the other two patients, it was close to the coronary sinus ostium.

Fig. 4A shows the ablation site in patient 1, which was between the ARAW showing manifest entrainment and the HB recording site, and 15 mm from the HB site toward the pacing site. The fluoroscopy image shows the ablation site remote from the HB recording site (Fig. 4B). The application of RF energy at this site resulted in the termination of tachycardia at 8 s after the initiation of ablation (Fig. 4C). A total of four RF applications resulted in the elimination of AT even after isoproterenol infusion.

A similar observation was made in the other four patients in whom ablation was done during AT, and AT was terminated in 6 ± 3 s (range, 3–11 s) after the RF application. Table 2 summarizes the results of ablation. AT was terminated or became noninducible by 1–3 RF applications, and the total application number was 3.7 per patient (range, 2–6). AT became no longer inducible even after isoproterenol infusion in all patients. The distance between the successful ablation site and the HB site was 19 ± 2 mm. The AH interval was 96 ± 12 ms before and 89 ± 7 ms after ablation ($p = 0.13$). In two of the patients, acceleration of tachycardia or cycle length irregularity was noted before the termination of tachycardia. Junctional rhythm or tachycardia was noted during ablation in none of the patients.

During a mean follow-up period of 11 ± 6 months (range, 5–19 months), no patients experienced recurrence of the tachycardia. There were no adverse events, including AV conduction disturbance.

Table 2
Results of ablation.

Ablation						AH interval (ms)	
Pt no.	Ablation site	Distance from the HB (mm)		No. of successful ablation (total application)	Acceleration during RF	Before ablation	After ablation
1	1 O'clock	15	During AT	1 (4)	+	104	92
2	3 O'clock	20	During AT	1 (2)	–	96	80
3	5 O'clock	20	During sinus	1 (2)		102	97
4	1 O'clock	18	During AT	3 (4)	+	110	98
5	1 O'clock	18	During AT	1 (4)	–	87	85
6	0 O'clock	20	During AT	3 (6)	–	76	84
Mean ± SD		19 ± 2				96 ± 12	89 ± 7

AH=atrio-His; AT=atrial tachycardia; HB=His bundle recording site; Pt no.=patient number; RF=radiofrequency.

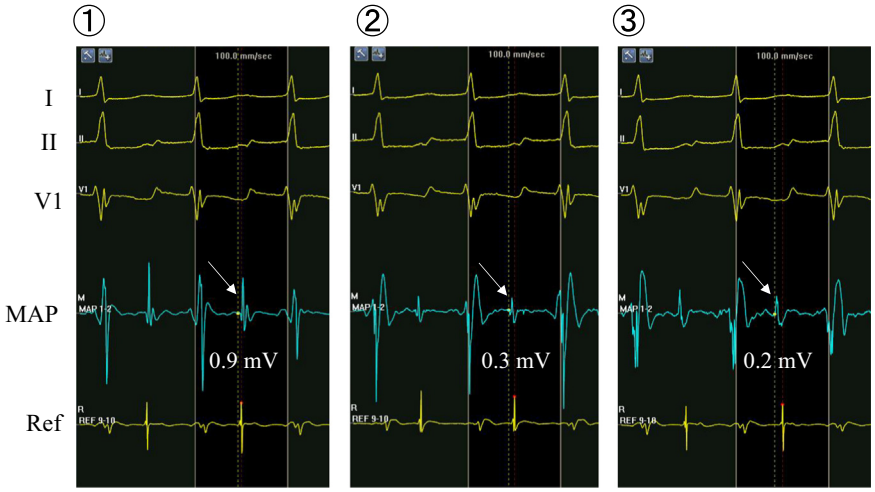

Fig. 5. Bipolar atrial electrograms (MAP) recorded at the three sites illustrated in Fig. 4A (numbers in the circle). Arrows indicate atrial electrograms. Ref=reference electrogram.

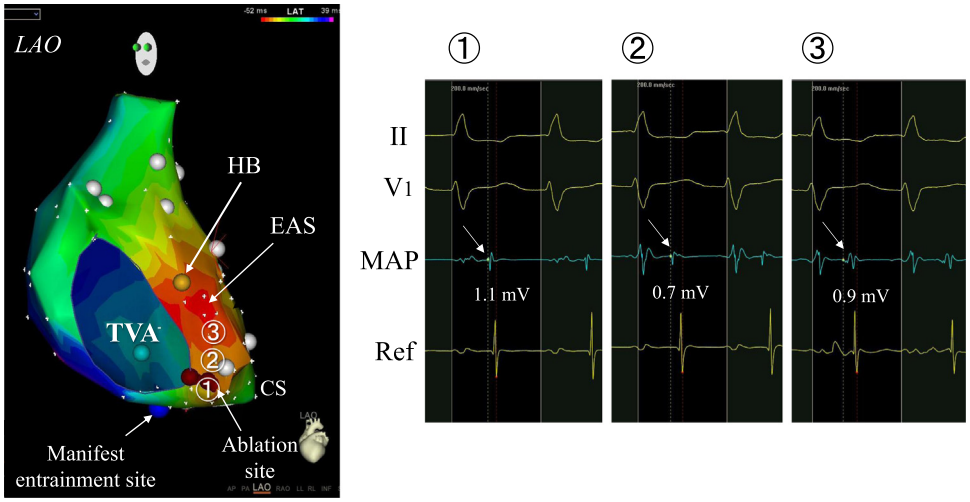

Fig. 6. *Panel A*, another example showing a focal pattern of activation during the tachycardia (patient 2). *Panel B*, bipolar atrial electrograms (MAP) recorded at the three sites illustrated in panel A (circled numbers). Arrows indicate atrial electrograms. EAS=earliest activation site; HB=His bundle recording site; Ref=reference electrogram; TVA=tricuspid valve area; LAO=left anterior oblique projection.

3.3. *Electrograms recorded at the successful ablation site*

Fig. 5 shows the bipolar atrial electrograms (MAP) recorded at the three sites illustrated in Fig. 4A (numbers in the circle). As indicated by arrows, no abnormal, fractionated electrograms were found. Fig. 6 shows another example with the manifest entrainment pacing site in the CTI-CS and the successful ablation site close to the coronary sinus ostium (patient 2). The atrial electrograms recorded at three sites (circled numbers in panel A) all showed normal configuration, and no fractionated potentials were noted (panel B). The other patients also had no abnormal electrograms at the successful ablation site and at the sites between the ablation site and the HB site or the EAS. The mean amplitude of the bipolar atrial electrogram at the successful ablation site was 0.8 ± 0.3 mV (range, 0.3–1.0 mV), and the ratio of

the atrial to the ventricular electrogram was 1.2 ± 0.9 (range, 0.3–2.3) for all patients.

4. Discussion

4.1. Major findings

With the use of CARTO mapping- and manifest entrainment-guided ablation strategy, ATP-AT originating from the AV node vicinity could be eliminated effectively and safely without causing AV conduction disturbance. We again demonstrated reentry with a slow conduction zone as a mechanism of tachycardia [7]; however, we failed to record any abnormal low-amplitude or fractionated potentials at the successful ablation sites. Although the mechanism for slow conduction remains unexplained, this study validated the ablation strategy in which RF application is targeted for the site remote from the EAS. This ablation strategy can be used as a first-line approach to ATP-AT originating from the AV node vicinity.

4.2. Mechanism of ATP-AT originating from the AV node vicinity

Iesaka et al. first reported AT mimicking the atypical form of AV nodal reentrant tachycardia and sensitive to a small dose of ATP [7], and suggested focal reentry without involvement of the AV nodal pathways. Yamabe et al. recently demonstrated that verapamil- or ATP-sensitive atrial tissue not in the AV conduction system forms the tachycardia reentry circuit of this AT [9]. Furthermore, by using a noncontact mapping system, it was demonstrated that this AT can be entrained by pacing at a site remote from the EAS, fulfilling the classic entrainment criteria [14]. This article first revealed the spatial relation of the ablation site and EAS, but did not demonstrate any direct evidence of a slow conduction zone. Thus, the anatomical localization of the reentry circuit of this AT still remains to be determined.

With the use of a more accurate contact mapping system (CARTO), we showed a focal activation pattern during ATP-AT in the successive six patients. As demonstrated previously [14], AT was entrained by the pacing from the ARAW or CTI-CS, fulfilling the present criteria for manifest entrainment. To demonstrate manifest entrainment, we carefully chose the pacing site from among the ARAW, upper and lower interatrial septum, and CTI-CS. Moreover, the pacing rate was set to be faster by only 5–10 beats/min than the AT rate, which resulted in the increase of the AT rate to the pacing rate. Accordingly, we demonstrated manifest entrainment from one site in all patients. Pacing at faster rates would result in the antidromic capture of the EAS such as that seen during pacing from a site distal to a slow conduction zone (Fig. 2).

Most important, we again demonstrated a long conduction interval during entrainment. As this AT could be eliminated by a few RF applications between the manifest entrainment pacing site and the EAS, the presence of a slow conduction zone is strongly suggested at least in this area. We therefore concentrated on finding any abnormal, low-amplitude, fractionated electrograms that allow slow conduction and small reentry mimicking focal tachycardia [16]. As shown in Figs. 5 and 6, however, we failed to record such abnormal potentials. Thus, the mechanism of this AT can be explained by reentry functionally, but still not anatomically even with a contact mapping system.

In the AV nodal reentrant tachycardia, the slow pathway serves as a slow conduction zone. However, there have been no reports demonstrating evidence for slow pathway activation during the tachycardia by using a conventional catheter electrode technique [18]. Even with the current technologies, recording potentials from the tissues with Ca channel-dependent conduction such as the AV node is not possible in the human heart. It should be remembered that AV node-like structures are present along the tricuspid annulus of the normal hearts as remnants of specialized AV ring tissue [19]. There are cells with AV nodal characteristics present in the superficial layer of the tricuspid annulus of porcine and canine hearts [20]. In fact, AT originating from the tricuspid annulus other than the AV node vicinity and with similar properties to the present ATP-AT has been reported [8,9,13]. In the present six patients, manifest entrainment was possible only from the ARAW (Fig. 1A) and CTI-CS (Fig. 6A), both of which were close to the tricuspid annulus, and the mean ratio of the atrial to the ventricular electrograms at the successful ablation site was 1.2. Thus, the AV node-like or transitional cell tissue is suggested to be present over the normal atrial myocardium in the tricuspid annulus, and serves as a slow conduction zone in the present ATP-AT. The acceleration of tachycardia during RF application seen in two patients also suggests the presence of AV node-like tissue. A technical inability to show slowly conducting impulse in this slow conduction zone possibly resulted in the focal activation pattern in the CARTO map.

4.3. Efficacy and safety of the present ablation strategy

A previous ablation strategy for ATP-AT originating from the AV node vicinity targeted the EAS during the tachycardia with a risk of AV conduction impairment. In fact, transient AV block was induced by this strategy [7]. Yamabe's strategy and ours target not the EAS but the site 19 ± 2 mm remote from the HB recording site (Fig. 4A, B). None of the patients showed AV conduction impairment due to the ablation, and the AH interval remained unchanged. After the ablation, AT was eliminated in all patients, and during the 11-month period, no patients experienced a recurrence of AT. Thus, the present manifest entrainment-guided ablation strategy is effective and safe.

Recently, Yamabe et al. found that verapamil- and ATP-sensitive AT originating from the tricuspid annulus other than the AV node vicinity can also be ablated at the site 10.4 mm remote from the EAS [21]. This AT and the present ATP-AT may form a single entity of AT with a slow conduction zone in the tricuspid annulus, as described above.

4.4. Limitations

To demonstrate manifest entrainment of AT, a stable, sustained tachycardia needs to be induced, and therefore, the present ablation strategy cannot be applied to unsustained or unstable tachycardia showing cycle length variation. Isoproterenol seems to be useful in stabilizing the tachycardia. The number of the study patients was small. We studied consecutive cases with sustained ATP-AT in which CARTO mapping and entrainment study was accomplished. The results were consistent among the cases and, therefore, the present results may be generalized. Manifest entrainment can show the relative location of the pacing site to a slow conduction zone, but cannot directly guide to the successful ablation site as concealed entrainment can. In fact, for eliminating AT, a mean of 3.7 RF applications were required. As there were no specific electrogram morphologies at the successful ablation, multiple RF applications may be necessary in some patients. We sought a site from which manifest entrainment was demonstrated in this study. If a site with manifest entrainment and with PPI close to the AT cycle length could be found, then the pacing site should be close to the entrance to the slow conduction zone. This might be associated with a reduced number of ablation for eliminating ATP-AT. Further studies are required.

5. Conclusions

ATP-AT with the EAS in the vicinity of the AV node is due to reentry with a slow conduction zone not demonstrable either by the conventional electrode catheter technique or by the CARTO system. Manifest entrainment-guided ablation strategy is effective and safe in the treatment of this ATP-AT.

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Conflict of interest

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None.

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